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Risky Alcohol Use and Age at Onset of Regular Alcohol Consumption in Relation to Frontal Lobe
Indices, Reward Sensitivity and Rash Impulsiveness

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Abstract

Objective: Excessive alcohol consumption has been linked to predisposing traits that may reflect frontal lobe functioning, an idea explored by the present study. **Method:** The study recruited 132 adults of both genders aged 18-68 who completed an online questionnaire battery. **Results:** Two aspects of impulsivity (reward sensitivity and rash impulsiveness), as well as the Disinhibition and Executive Dysfunction indices of the Frontal Systems Behavior Scale (FrSBe), were associated with risky drinking as defined by the Alcohol Use Disorders Identification Test (AUDIT). Reward sensitivity (SR) partially mediated the association between Disinhibition and AUDIT. Higher SR predicted younger age at onset of weekly drinking (AOD). Parental alcoholism as measured by the Children of Alcoholics Screening Test was also associated with younger AOD and riskier drinking. By contrast, sensitivity to punishment (SP) was associated with later AOD in older participants, and with lower alcohol consumption in younger participants. **Conclusions:** The present findings suggest that several interrelated and heritable personality and neurobehavioral traits may promote earlier as well as riskier alcohol consumption.

Key Words: Alcohol ; Alcohol Abuse ; Frontal Lobe ; Impulsivity ; Reward Dependence

Research on the etiology of problematic alcohol use has invoked neurobiological and personality variables that preceded alcohol exposure as well as neurobehavioral deficits attributed to the duration and severity of exposure (Kreek, Nielsen, Butelman, & LaForge, 2005; Lyvers, 2000; Simons, Gaher, Correia, Hansen, & Christopher, 2005; Varma, Basu, Malhotra, Sharma, & Mattoo, 1994; Verdejo-García, Rivas-Pérez, López-Torrecillas, & Pérez-García, 2006; Volkow & Li, 2004). Factors said to promote alcoholism include an inherited mild dysfunction of the prefrontal cortex (Spinella, 2003), personality traits such as impulsivity and neuroticism (Hair & Hampson, 2006; Kambouropoulos & Staiger, 2007; Schmidt, Buckner, & Keough, 2007; Zuckerman & Kuhlman, 2000), an early onset age for excessive alcohol use at a time of cognitive vulnerability (Crews, He, & Hodge, 2007; Pitkänen, Lyyra, & Pulkkinen, 2005) and the cumulative effects of prolonged high alcohol exposure (Kubota et al., 2001; Lyvers, 2000).

The “frontal lobe” hypothesis of addictions suggests that impairment of the capacity to properly evaluate rewards and punishments and to regulate and inhibit impulsive behavior are critical factors promoting the onset and maintenance of compulsive substance use (Lyvers, 2000). The prefrontal cortex regulates limbic and parietal regions (Phan et al., 2005) in a synergistic process which is compromised by excessive alcohol usage, as demonstrated in the effects of acute and chronic intoxication (Giancola, 2000; Rose & Duka, 2007; Sullivan & Pfefferbaum, 2005; Sullivan, Rosenbloom, & Pfefferbaum, 2000). The orbitofrontal area is particularly implicated as it is involved in responding to reinforcers, assessing risks and inhibiting inappropriate behavioral responses (Beer, Knight, & D'Esposito, 2006; Berlin, Rolls, & Kischka, 2004; Hornak et al., 2004; Rolls, 2004). Early onset heavy drinking has been implicated in possible alcohol induced changes to the brain of adolescents (Crews et al., 2007; Monti et al., 2005; Verdejo-García et al., 2006; Volkow & Li, 2004). Another possibility however is that mild prefrontal cortical dysfunction may predate the onset of excessive alcohol use in young people as a genetic predisposition (Agrawal & Lynskey, 2008; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1973; Lyvers, Czerczyk, Follent, & Lodge, 2009; Yacubian et al., 2007). Consistent with a genetic interpretation, early onset age of

regular drinking is associated with trait indices of reward sensitivity (Lyvers et al.) and is also a strong predictor of life-time alcohol misuse (Pitkänen et al., 2005). Further, early onset alcoholism is associated with trait impulsivity (Pardo, Aguilar, Molinuevo, & Torrubia, 2007) and familial alcoholism (Dom et al., 2006; Varma et al., 1994). A predisposition to excessive alcohol use in adolescents and adults may reflect interactions between the limbic system and orbitofrontal cortex (Enoch & Goldman, 2002) that manifest as reduced behavioral inhibition and increased impulsivity (Berlin et al., 2004; Franken, van Strien, Nijs, & Muris, 2008) prior to alcohol exposure.

Impulsivity, risk taking and rash decision making in anticipation of reward have been linked to orbitofrontal and dopaminergic system dysfunction (Berlin et al., 2004; Elliott & Deakin, 2005; Franken et al., 2008; Schoenbaum & Shaham, 2008; Yacubian et al., 2007). Dopaminergic neurons promote approach behavior when activated by cues predicting reward and pause in their firing when an expected reward is not received (Schultz, Tremblay, & Hollerman, 2000; Schulz, 2001). An overactive sensitivity to reward involves reduced ability to evaluate the implications of negative outcomes, such that negative emotions aroused by errors or loss of reward are less able to influence decision making (Beer et al., 2006; Hornak et al., 2004). Failure to recognize the loss of reward, and elevated anticipation irrespective of reward probability and magnitude, have been demonstrated in patients with orbitofrontal damage (Hornak et al., 2004). Impulsive behavior and high reward sensitivity have also been associated with problematic drinking and other forms of substance abuse (Dawe, Gullo, & Loxton, 2004; Dawe & Loxton, 2004; Hanson, Luciana, & Sullwold, 2008; Lyvers et al., 2009; O'Connor & Colder, 2005; Simons et al., 2005),

Both self-report measures (Dom et al., 2006) and objective laboratory tasks (Bjork et al., 2004) have indicated a negative relationship between impulsivity and age of onset of drinking. Dawe et al. (2004) proposed that impulsivity has two distinct dimensions: reward sensitivity (SR), or the degree to which behavior tends to be motivated by the prospect of positive reinforcement, and rash impulsiveness, or difficulties with the inhibition of behavior. They suggest that the former can be measured by the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ;

Torrubia, Ávilab, Moltób, & Caseras, 2001) and the latter by the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995), suggestions implemented in the present study. In Dawe et al.'s model, SR was proposed to play a major role in the onset of regular substance use, whereas rash impulsiveness was said to promote ongoing excessive or problematic substance use reflecting orbitofrontal dysfunction and associated disinhibition of behavior (Dawe et al., 2004; Dawe, Loxton, Gullo, & Staiger, 2007; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Ketzenberger & Forrest, 2000).

A few studies have examined alcohol consumption in relation to measures of SR based on Gray's Reinforcement Sensitivity Theory (RST), such as the BIS/BAS scales (Carver & White, 1994) where the BAS (Behavioral Activation System) Scale is an index of SR and the BIS (Behavioral Inhibition System) Scale is an index of sensitivity to punishment (SP) (Corr, 2002). Loxton and Dawe (2001) reported that BAS but not BIS scores were related to alcohol consumption as measured by the Alcohol Use Disorders Identification Test (AUDIT; Babor, de la Fuente, Saunders & Grant, 1992), and Pardo et al. (2007) reported a positive association between BAS scores and alcohol consumption as well as a negative association between BAS scores and age at first alcohol drink. As age at first drink is likely to reflect situational factors, the present study asked for the age at which regular (i.e., weekly) drinking was initiated, because this was expected to be more sensitive to individual differences in SR and was in line with other recent findings (Lyvers et al., 2009) as well as theory (Dawe et al., 2004). Although a direct relationship between anxiety or SP and alcohol use has not been conclusively established (Loxton & Dawe, 2001), Feil and Hasking (2008) suggested that anxiety and stress can play an important role in the development of excessive drinking. According to Kambouropoulos and Staiger (2004) individuals with high SP drink to alleviate environmentally aroused aversive states such as anxiety. Alcohol may thus be used by some to dampen an over-reactive aversive motivational system (Fowles, 1987). On the other hand, anxiety is conjectured to serve as a protective factor against disinhibited, potentially dangerous activities and behaviors that could lead to early mortality in adolescence and early adulthood (Lee,

Wadsworth, & Hotopf, 2006). Thus high levels of SP might be expected to protect against alcohol problems in young adults, despite the possibility of later onset of excessive drinking to alleviate negative states such as anxiety (Kambouropoulos & Staiger, 2004). This idea was one of several investigated in the present study.

Self-report questionnaires are commonly used to assess personality traits and are increasingly regarded as an efficient means of examining hypotheses about neurobiological functioning (Verdejo-Garcia, Lawrence & Clark, 2008). The Frontal Systems Behavior Scale (FrSBe; Grace & Malloy, 2001) has been shown to identify dysfunction as well as subtle gradations of functioning associated with three frontal systems (dorsolateral, orbitofrontal, anterior cingulate) in both clinical and non-clinical samples (Spinella, 2007a). A previous study by Lyvers et al. (2009) found that FrSBe Disinhibition scores (designed to assess orbitofrontal dysfunction) and SPSRQ-SR scores were both positively correlated with scores on the AUDIT, where higher scores indicate heavier alcohol consumption and riskier drinking. Further, SR scores were negatively related to age at onset of regular alcohol use, and high risk drinkers (as defined by AUDIT) had higher FrSBe Disinhibition and SR scores compared to low risk drinkers. These findings cannot distinguish between characteristics which developed since the onset of alcohol use and inherent characteristics which preceded alcohol use (Verdejo-García et al.). However, as the findings of Lyvers et al. were obtained in a young adult sample of university undergraduates aged 18-25 years, the authors suggested that the links between SR, Disinhibition and AUDIT most likely reflected trait factors predisposing to earlier and heavier alcohol consumption rather than neurobiological effects of chronic heavy drinking. The present study recruited participants across a much broader age range, as arguably the factors promoting heavier alcohol consumption in older adults might vary from those in younger age groups (Schmidt et al., 2007; Varma et al., 1994; Welte & Mirand, 1995). Further, the present study included the BIS-11 as an index of rash impulsiveness in addition to the SPSRQ measure of SR as per Dawe and Loxton's (2004) notion of two separable components of impulsivity. Parental alcoholism as assessed by the Children of Alcoholics Screening Test (CAST-

6; Hodgins, Maticka-Tyndale, El-Guebaly & West, 1993) was also examined in relation to SR, Disinhibition, BIS-11, AUDIT and age at onset of regular drinking (AOD), with the expectation that trait factors related to heavier and earlier consumption (such as Disinhibition and SR) would be associated with parental alcoholism in line with presumed genetic risk factors for alcohol problems.

Method

Participants

The 132 participants were 87 females and 45 males; ages ranged 18 to 68 years ($M = 36.06$, $SD = 14.65$), of whom 35% fell within the 18 to 25 year age group (young adults). All participants were at least occasional alcohol drinkers (note that the legal drinking age in Australia is 18). The sample included 40 students from the university's undergraduate psychology participant pool who participated for course credit, and 92 members of the local community recruited through local high school teachers (snowball method; no incentive offered). In terms of education level, 80% of the sample reported that they had completed year 12 or higher. Only 1.5% of the sample said they were regular users of illicit drugs (defined as using more than three times per month), and 20% indicated they were current tobacco smokers, consistent with Australian Institute of Health and Welfare (AIHW, 2008) national averages. The overall sample size was adequate for multiple regression as recommended by Tabachnik and Fidell (2007).

Materials

A commercial online survey program, *Survey Monkey*, was used to present the questionnaires described below via the internet.

The Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 1992) is composed of 10 questions, including 3 quantity/frequency questions (e.g., "How often do you have a drink containing alcohol?"), 3 dependence-related items (e.g., "How often during the last year have you failed to do what was normally expected of you because of drinking?"), and 4 alcohol-related consequences or harm (e.g., "Have you or someone else been injured because of your drinking?"). AUDIT questions are scored from 0 to 4, with an overall score ranging from 0-40. The suggested

cut-offs for Hazardous and Harmful drinking are 8 and 16, respectively. Internal consistency (Cronbach's alpha) and test-retest reliability are consistently reported to be high (Bergman & Källmén, 2002; Dybek et al., 2006; Kane, Loxton, Staiger, & Dawe, 2004; Pal, Jena, & Yadav, 2004; Rubin et al., 2006). Convergent validity has been supported by correlations with the Michigan Alcoholism Screening Test of up to .97 (Pal et al., 2004).

The *Frontal Systems Behavior Scale* (FrSBe; Grace & Malloy, 2001) Self-Rating form is a 46-item questionnaire assessing behavioral evidence of dysfunction in three major prefrontal-subcortical systems of the brain. There are three subscales: Apathy (poor initiation, reduced drive and interest), designed to assess anterior cingulate dysfunction; Disinhibition (distractibility, problems with inhibition, socially inappropriate behavior), designed to assess orbitofrontal dysfunction; and Executive Dysfunction (difficulties with planning, sequencing, working memory, and mental flexibility), designed to assess dorsolateral prefrontal dysfunction. The standard version of the Self Rating form of the FrSBe aims to measure behavioral change by obtaining pre-and post-lesion ratings. For the purposes of this study and in keeping with previous research (Lyvers et al., 2009; Spinella, 2003; Verdejo-García et al., 2006) only overall scores in present time were obtained. Items are rated on a 5 point Likert scale (1=almost never, 2= seldom, 3= sometimes, 4= frequently, 5= almost always). The first 32 items represent deficits and are rated accordingly, with the final 14 positively stated items reverse scored. The magnitude of the score on each subscale indicates the degree of impairment. Factor analyses of the FrSBe in several neurological populations have supported the construct validity of the subscales (Stout, Ready, Grace, Malloy, & Paulsen, 2003). Evidence also supports reliability (Velligan, 2002) with internal consistency from .88 to .91 and three month test-retest reliability of .78. Diagnostic validity has been confirmed for detecting graduated degrees of symptoms of frontal lobe functioning in various clinical samples (Chiaravallotti & DeLuca, 2003; Velligan, 2002), in substance use and abuse populations (Spinella, 2003) and in healthy individuals (Spinella, 2007a).

The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia et al., 2001) is a 48 yes-no response item questionnaire consisting of two scales: Sensitivity to Punishment (SP; 24 items, e.g. “Are you often afraid of new or unexpected situations?”), and Sensitivity to Reward (SR; 24 items, e.g. “Do you sometimes do things for quick gains?”). Dichotomous responses of either “yes” (1) or “no” (0) yield a score for each scale which is a summation of all affirmative responses. The magnitude of the score indicates the level of SR and SP. Both scales demonstrate acceptable levels of internal consistency and three month test–retest reliability (O'Connor & Colder, 2005; Torrubia et al., 2001). Construct validity has been demonstrated in a two factor solution (Caci, Deschaux, & Bayle, 2007; O'Connor, Colder, & Hawk, 2004). SR has been found to be positively related to extraversion ($r = .48$) and Gray's BAS scale ($r = .43$), and SP related to neuroticism ($r = .70$) and Gray's BIS scale ($r = .50$) (Sava & Sperneac, 2006), consistent with the theoretical underpinnings of the SPSRQ.

The *Barratt Impulsiveness Scale* (BIS-11; Patton et al., 1995) is a 30-item self-report questionnaire designed to measure trait impulsivity. Factor analysis in a clinical population (Patton et al., 1995) and later replicated in a non-clinical, community population (Spinella, 2007b) demonstrated the construct validity of three separate impulsivity subscales: (a) non-planning impulsivity or the inability to plan and think carefully; 11 items, e.g. “I plan tasks carefully” (reverse scored item), (b) motor impulsivity or acting on the spur of the moment; 11 items, e.g. “I do things without thinking,” and (c) attentional impulsivity or the inability to focus on the task at hand; 8 items, e.g. “I don't pay attention.” Items are rated on a four-point Likert scale ranging from “rarely/never” to “almost always.” Individual items are summed to create an overall score, with higher scores representing greater levels of impulsiveness. Research has established that the BIS-11 has strong psychometric properties in both psychiatric (Patton et al., 1995) and non-clinical populations (Spinella, 2007a) with reliability coefficients from .79 to .83 and test-retest stability over one year of .60 (Luengo, Carrillo-de-la-Pena, & Otero, 1991). Convergent validity was established with neuropsychological measures that have demonstrated sensitivity to orbitofrontal

prefrontal dysfunction (the Go/No-Go Test, $r = .39$, and the Antisaccade Test, $r = .48$; Spinella, 2004) as well as related subscales of the FrSBe (Executive Dysfunction, $r = .70$; Disinhibition, $r = .60$; Spinella, 2007b).

The *Children of Alcoholics Screening Test-6* (CAST-6) was developed by Hodgins et al. (1993) as a short version of the CAST, a widely used 30 item yes-no scale designed to assess perceptions of parental impairment due to alcohol to identify adult children of alcoholics. The CAST-6 consists of 6 items (e.g., “Did you ever wish that a parent would stop drinking?”). Dichotomous responses of either “yes” (1) or “no” (0) produce a score which is a summation of all affirmative responses. A score of 3 or more indicates a probable child of an alcoholic parent. The CAST-6 was derived from 6 items which loaded highest across clinical and non-clinical samples after independent principle components analysis (Hodgins et al.). Internal consistency and test-retest stability are high, and CAST-6 scores are highly correlated with scores on the 30-item CAST ($r = .93$; Hodgins & Shimp, 1995).

A *Demographic Questionnaire* asked participants to provide personal information on age, gender, years of education, current age, the age at which they started drinking weekly (AOD), illicit drug use and smoking.

Procedure

After obtaining ethical clearance from the Bond University Human Research Ethics Committee (BUHREC), recruitment of university students was undertaken via poster advertisement and sign-up sheets which directed interested participants to a room on campus with computers for completion of the online questionnaire battery. The 40 undergraduate university students participated for course credit in introductory psychology classes. Community participants were recruited through emailed invitation to local high school teachers. Each community volunteer was invited to recruit further participants from their own personal networks. No incentive was provided for community participants. All participants were required to be at least 18 years of age. Those who volunteered were provided access to the online questionnaire site. A commercial online survey

program, Survey Monkey, was used to conduct the study. The questionnaires were completed individually online and submitted through a temporary link. Access to the questionnaires was only made available after the explanatory statement had been read and online consent had been indicated. Completion took approximately 30 minutes. All participants were de-identified by the survey program which had been designed so that answers could not be tracked to computer ISP addresses or individuals as per ethics committee requirements. Answers were automatically coded and collated into an anonymous data pool. The data were analysed with the SPSS 16 Graduate Pack statistical package.

Results

Analyses included bivariate correlations, between-subjects multivariate analyses of covariance (MANCOVA), standard multiple regressions and pathway analysis. The variables were examined separately for missing values and fit between distributions and assumptions. Six cases with missing data in the Years of Education variable were replaced with the mean. Boxplots identified several moderate outliers. Preliminary analysis for univariate outliers using z scores found no extreme outliers although several cases recorded standardised scores in excess of the ± 3.29 ($p < .001$) recommended by Tabachnik and Fidell (2007, p.73). One of these cases was also identified in the residual scatterplot and through Casewise Diagnostics with a standardised residual approaching 3.3. Tabachnik and Fidell (2007, p.73) advise that a few scores in excess of the recommended standardised residual are to be expected with a large sample. With the use of a $p < .001$ criterion for Mahalanobis distance this case was also identified as a multivariate outlier. Investigation of survey responses identified all outliers as being indicative of the intended population and appropriate to the sample. The size of the data set ($n = 132$) was deemed to allow retention of all cases with minimal impact. Assumptions concerning linearity and multicollinearity were investigated and met.

Normality in the scatterplot and the normality line for standardized residuals were deemed acceptable for AUDIT score as a dependent variable but indicated some departure from normality

for AOD as expected. Deviations from normality in the AUDIT scores (skewness = 1.5) were judged to not make a substantive impact on analyses due to the sample size (Tabachnik & Fidell, 2007, p.80). Of the present sample 57.58% reported Low Risk drinking as defined by AUDIT ($n = 76$), whereas one-third were defined as Hazardous drinkers ($n = 44$) and 9% scored in the Harmful range ($n = 12$). These results were consistent with the Australian Institute of Health and Welfare (AIHW, 2008) national average for alcohol consumption. The age at which participants started drinking weekly (AOD) ranged from 13 to 48 years ($M = 19.18$, $SD = 6.13$).

Of the 132 participants only 34 were classified as children of alcoholics by the CAST-6, all of whom reported an early AOD (before the age of 25). The mean AOD for children of alcoholics ($M = 17.06$ years, $SD = 1.89$) was significantly earlier than for other participants ($M = 19.92$ years, $SD = 6.89$), $t(126) = 3.77$, $p < .0001$ (unequal variances assumed). Of the participants classed as children of alcoholics, 56% drank at a risky level (Hazardous or Harmful) compared to 38% in the rest of the sample. The mean AUDIT score for children of alcoholics was above the AUDIT cutoff score of 8 ($M = 9.41$, $SD = 6.67$) which separates Low Risk from Hazardous drinkers, whereas the mean for other participants was below 8 ($M = 6.91$, $SD = 5.41$). An independent t-test indicated that AUDIT scores were significantly higher for children of alcoholics than for other participants, $t(130) = 2.17$, $p = .03$.

Intercorrelations were calculated among Years of Education, Age, AOD, CAST-6, AUDIT, FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ SP and SR, and BIS-11. These intercorrelations are shown in Table 1. The important correlations to note in Table 1 are (1) the significant positive correlations of AUDIT with FrSBe Disinhibition and Executive Dysfunction, SPSRQ SR, BIS-11 and CAST-6, and (2) the significant negative correlations of AOD with SR and CAST-6 but a positive correlation with SP. When correlations were conducted separately for younger (18-25 years) and older (26-68 years) participants, most of the significant correlations were very similar; i.e., in both age groups, AUDIT scores were significantly positively correlated with FrSBe Disinhibition and Executive Dysfunction as well as with SPSRQ SR and BIS-11. However,

AOD was positively correlated with SP in the older age group, $r(85) = .39, p < .0001$, but not the younger age group, $r(47) = .13, p = .38$; and CAST was positively correlated with AUDIT in the older age group, $r(85) = .36, p < .001$, but not in the younger age group, $r(47) = .04$, n.s.

A standard multiple regression was performed to explore influences of trait variables of interest on AOD. The regression was performed with AOD as the criterion and seven predictor variables: CAST, FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ SP and SR, and BIS-11. Table 2 displays the unstandardised regression coefficients (B), standardised regression coefficients (β), semi-partial correlations (sr^2), R^2 and adjusted R^2 . R for regression was significant, $F(7,124) = 4.86, p < .001$. The adjusted R^2 indicated that 17% of the variability in AOD was predicted by the seven factors. Although the CAST and SR both made a significant negative contribution, SP made the strongest unique contribution to AOD (see Table 2).

The hypothesis that BIS-11, SPSRQ SR, and FrSBe Disinhibition and Executive Dysfunction would vary according to AUDIT Risk Group (Low Risk, High Risk) was investigated through a 2 (Gender) x 2 (AUDIT levels) between-groups MANCOVA performed on all three FrSBe scales, the SPSRQ scales and BIS-11. Age was entered as a covariate given its relationship to most of the other variables (see Table 1). The High Risk group included both Hazardous and Harmful drinkers as defined by AUDIT. The assumption of homogeneity of variance-covariance matrices (Box's M) was not significant, indicating no violation. As the Levene's test was significant for SP and BIS-11, a more stringent alpha level was set ($p < .01$) (Tabachnik & Fidell, 2007). Pillai's trace indicated that the combined dependent variables were significantly affected only by the covariate age and by AUDIT risk level, $F(6, 122) = 5.17, p < .0001$, observed power = .99. There was no effect of Gender and no interaction. When the unique effects of the AUDIT grouping variable on the dependent variables were considered, all variables except Apathy and SP were significant: Disinhibition, $F(1, 127) = 24.89, p < .0001$, observed power = 1.00; Executive Dysfunction, $F(1, 127) = 7.36, p < .01$, observed power = .77; BIS-11, $F(1, 127) = 9.46, p < .01$,

observed power = .86; SR, $F(1, 127) = 6.45, p < .01$, observed power = .71. The AUDIT Risk Group means are shown in Table 3.

These relationships were further investigated using AUDIT as a continuous variable. A standard multiple regression was performed with total AUDIT scores as the criterion and six predictor variables: FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ SP and SR, and BIS-11. Table 4 displays the standardised regression coefficients (B), standardised regression coefficients (β), the semi-partial correlations (Sr^2), R^2 and adjusted R^2 . R for regression was significantly different from zero, $F(6, 125) = 11.91, p < .001$. The adjusted R^2 indicated that 33% of the variability in AUDIT was predicted by the six factors. Disinhibition made the strongest unique contribution when the variance of all other variables was controlled (see Table 4).

Disinhibition was significantly associated with AUDIT risk in the MANCOVA and was also a significant predictor of AUDIT score in the regression. Both BIS-11 and SR had a significant association with AUDIT risk in the MANCOVA but only SR was a significant predictor of AUDIT in the regression. These relationships were further investigated via a simple path analysis to determine the extent to which the influence of Disinhibition on AUDIT was mediated by either component of impulsivity (SR or BIS-11). With a significant relationship between Disinhibition and the AUDIT confirmed, BIS-11 and SR were regressed on Disinhibition followed by the regression of all three predictors (BIS-11, SR and Disinhibition) onto AUDIT. Exploratory regression with a centered interaction term for each proved nonsignificant, ruling out moderation. Figure 1 shows the path coefficients and the error variance. Although the Sobel test statistic for SR was significant ($t = 2.07, p < .05$), indicating convincing evidence of a strong mediation pathway, the relationship change between Disinhibition and AUDIT ($\beta = .52$ to $\beta = .39$) indicated only partial mediation. The path between Disinhibition and AUDIT, although weaker, was still significant (see Figure 1). The path coefficient between BIS-11 and AUDIT was nonsignificant ($\beta = .04$) and the Sobel test was nonsignificant ($t = 0.45, ns$). BIS-11 thus contributed little to prediction of AUDIT and did not mediate the extent to which Disinhibition determined AUDIT scores.

Discussion

The present findings are consistent with hypothesized links between prefrontal cortex dysfunction, reward sensitivity, rash impulsiveness and alcohol related risk. Approximately one third of the variation in AUDIT scores in the current sample was attributable to the measured personality and neurobehavioral factors.

Previous research indicated that impairment of orbitofrontal inhibitory processes is behaviorally manifested by disinhibition and impulsivity (Berlin et al., 2004; Elliott & Deakin, 2005; Franken et al., 2008; Horn et al., 2003; Schoenbaum & Shaham, 2008; Simons et al., 2005; Spinella, 2003). Consistent with this idea, in the present study the self-report index of orbitofrontal dysfunction - FrSBe Disinhibition score - was moderately related to both SR and BIS-11 (indices of sensitivity to reward and rash impulsiveness components of impulsivity, respectively; Dawe et al., 2004), and all three variables were moderately related to AUDIT. A similar pattern was evident for FrSBe Executive Dysfunction scores, a scale designed to reflect dysfunction of the dorsolateral prefrontal cortex (Grace & Malloy, 2001). Of the three FrSBe scales designed to detect signs of frontal lobe dysfunction in everyday life, only the Apathy scale, which was designed to reflect anterior cingulate dysfunction, was unrelated to AUDIT (although Apathy too was significantly related to SP, SR and BIS-11). The present findings thus support the hypothesized links between mild dysfunction of prefrontal (especially orbitofrontal) cortex, sensitivity to reward, rash impulsiveness and alcohol related risk. Further, CAST-6 scores were significantly positively related to AUDIT, FrSBe Disinhibition and Executive Dysfunction, SR and BIS-11, and were negatively related to AOD (such that higher CAST-6 scores were associated with earlier age of onset of weekly alcohol use). Although these associations were not strong, they are consistent with the hypothesis that inherently poorer frontal lobe functioning, and higher sensitivity to reward and rash impulsiveness, may reflect heritable risk factors for risky or harmful alcohol consumption.

In patients with orbitofrontal damage, disinhibition is associated with impulsivity and elevated sensitivity to reward (Hornak et al., 2004). In the present study such an association was

supported in a non-clinical sample, with Disinhibition scores related to both SR and BIS-11 scores. The primary predictor of AUDIT was Disinhibition, and the strength of the relationship of Disinhibition with AUDIT was partially reduced by the filtering effect of SR as a mediator. The association between Disinhibition and rash impulsiveness (BIS-11) was also supported in this study; however, a pathway between Disinhibition, rash impulsiveness and alcohol related risk, which had been theoretically proposed and supported in previous research (Dawe et al., 2004), was not supported in the present study. Contrary to expectation, BIS-11 contributed little to prediction of AUDIT and did not mediate the extent to which Disinhibition influenced AUDIT scores. This may have been because the current sample consisted predominantly of low to medium risk drinkers, with only 12 participants reporting Harmful drinking as defined by AUDIT. Alcoholics have been found to exhibit high levels of rash impulsiveness (Ketzenberger & Forrest, 2000) but the present non-clinical sample was not characterised by drinking at an alcoholic level. Nevertheless both BIS-11 and Executive Dysfunction scores did significantly differentiate Harmful drinkers from the other groups in the present study. The high positive correlation between BIS-11 and Executive Dysfunction, and the fact that BIS-11 and Executive Dysfunction significantly differentiated Harmful drinkers from the other groups, is consistent with a theoretical link between impaired executive control, rash impulsiveness and problematic drinking patterns (Lyvers, 2000). Rash impulsiveness (BIS-11) distinguished Harmful from Hazardous drinkers (as defined by AUDIT) in the present study, whereas SR differentiated Low Risk drinkers from the higher risk levels. This pattern of findings is consistent with the notion that each component of impulsivity has a particular role in drinking behavior, with SR promoting change from Low Risk to Hazardous drinking, and rash impulsiveness promoting and maintaining Harmful drinking (Dawe et al., 2004).

The inverse relationship between SR and the age at which an individual started drinking regularly (AOD) suggests that over-responsiveness to reward contingencies rather than rash impulsiveness influences the drinking-related choices made at younger age levels (Crews et al., 2007; Loxton & Dawe, 2001; Monti et al., 2005; Pardo et al., 2007; Volkow & Li, 2004).

Conversely the positive relationship between AOD and SP implies a possible protective influence such that trait anxiety may promote delay of regular alcohol use (Feil & Hasking, 2008; Varma et al., 1994). Of all variables, SP made the strongest unique contribution to AOD in the overall sample. However when younger (18-25) and older (26-68) adults were examined separately, an association between SP and AOD was only found in the older adults, suggesting cohort related differences. The negative relationship between the CAST-6 and AOD, and the positive relationship of CAST-6 with AUDIT, supports research suggesting a genetic predisposition to heavier and earlier alcohol consumption, possibly involving impulsivity and reward sensitivity as heritable traits (Agrawal & Lynskey, 2008; Dawe et al., 2007; Dom et al., 2006; Goodwin et al., 1973; Kambouropoulos & Staiger, 2004; Varma et al., 1994). Thus CAST-6 was significantly, albeit weakly, related to SR and BIS-11 as well as Disinhibition and Executive Dysfunction.

A limitation of the present study was that although the sample included a broad age range, it was predominantly female and relatively well-educated. In order to confirm and extend the present findings, further research is required in a larger and broader sample that exhibits parameters more consistent with the general population. Ideally, longitudinal studies should be conducted in adolescents to establish whether the traits associated with alcohol use, as reported above, truly reflect an inherent predisposition to earlier and heavier alcohol consumption rather than consequences thereof.

In conclusion, the present findings are consistent with the hypothesis that a group of heritable, interrelated personality and neurobehavioral traits promote earlier and riskier alcohol consumption, and that such traits may to some extent reflect frontal lobe (particularly orbitofrontal) functioning. The popular notion of an “addictive personality” may have little foundation, but the role of premorbid neurobehavioral traits in substance abuse risk is a topic that demands extensive research attention.

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Table 1

Intercorrelations among Years of Education (Educ), Age, Age of Onset of weekly Drinking (AOD), CAST-6 (CAST), AUDIT (AUD), FrSBe-Apathy (Apath), FrSBE-Disinhibition (Disin), FrSBe-Executive Dysfunction (Exec), SP and SR from the SPSRQ, and BIS-11.

	Educ	Age	AOD	CAST	AUD	Apath	Disin	Exec	SP	SR
Age	.017									
AOD	.138	.418**								
CAST	-.069	-.195*	-.186*							
AUD	-.023	-.245**	-.159	.237**						
Apath	-.055	-.268**	-.070	.098	.121					
Disin	.018	-.369**	-.118	.245**	.519**	.527**				
Exec	-.109	-.312**	-.119	.206*	.334**	.736**	.754**			
SP	.017	-.138	.245**	.087	-.025	.444**	.187*	.335**		
SR	-.006	-.523**	-.198*	.190*	.416**	.407**	.520**	.443**	.396**	
BIS-11	-.184*	-.261**	-.075	.183*	.372**	.446**	.660**	.739**	.152	.368**

** p < .01 * p < .05

Table 2

Standard multiple regression of the CAST-6, FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ Sensitivity to Punishment and Sensitivity to Reward, and BIS-11 Impulsiveness on Age of Onset of weekly Drinking.

Variables	B	Std. Err	β	Sr ²
CAST-6	-.495	.248	-.165*	-.03
Apathy	-.090	.129	-.088	-.003
Disinhibition	.130	.117	.148	.007
Executive Dysfunction	-.165	.132	-.222	-.01
Sensitivity to Punishment	.477	.098	.459***	.15
Sensitivity to Reward	-.479	.144	-.334**	-.08
Impulsiveness	.068	.075	.113	.005
				$R^2 = .216$
				Adjusted $R^2 = .171$
				$R = .46^{***}$
*** $p < .001$ ** $p < .01$ * $p = < .05$				

Table 3

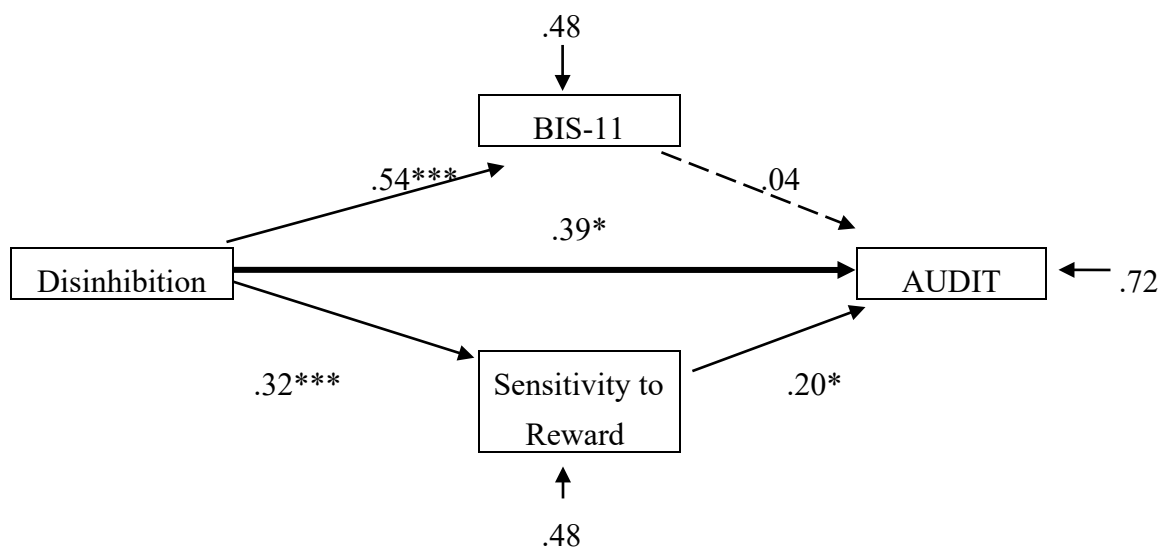
Means (SD) for FrSBe Disinhibition (Disin), FrSBe Executive Dysfunction (Exec), SPSRQ Sensitivity to Reward (SR), and Impulsiveness (BIS-11) as a function of AUDIT Risk Group.

AUDIT Risk Group:	Low Risk (<i>n</i> = 76)	High Risk (<i>n</i> = 56)
Disin	26.72 (5.74)	33.34 (6.79)
Exec	33.68 (7.79)	38.32 (8.17)
SR	9.45 (4.23)	12.48 (3.72)
BIS-11	58.07 (8.62)	64.05 (11.32)

Table 4

Standard multiple regression of FrSBe Apathy, Disinhibition and Executive Dysfunction, SPSRQ Sensitivity to Punishment and Sensitivity to Reward, and BIS-11 Impulsiveness on the AUDIT.

Variables	B	Std. Err	β	Sr ²
Apathy	-.212	.110	-.281	-.002
Disinhibition	.365	.100	.437***	.07
Executive Dysfunction	.021	.113	.030	.0001
Sensitivity to Punishment	-.146	.084	-.148	-.06
Sensitivity to Reward	.403	.122	.295**	.06
Impulsiveness	.041	.064	.072	.002
				$R^2 = .364$
				Adjusted $R^2 = .333$
				$R = .603^{***}$
*** $p < .001$		** $p < .01$		



*** $p < .001$ * $p < .05$

Figure 1. Path coefficients for the associations between FrSBe Disinhibition and AUDIT, with confirmed partial mediation by SPSRQ Sensitivity to Reward and unconfirmed mediation by BIS-11. Standardised betas are shown for all paths.